Dental Abnormalities Associated with Cleft Lip and/or Palate

RONALD E. JORDAN, D.D.S., M.S.D.
BERTRAM S. KRAUS, Ph.D.
C. MARSHALL NEPTUNE, M.D., D.D.S.
Pittsburgh, Pennsylvania

For a number of years Kraus and his associates have been engaged in studies directed toward defining normal patterns of dental crown development and morphology. The results were brought together in a book, *The Human Dentition Before Birth,* by Kraus and Jordan (*16*). One of the main purposes underlining this research was to set up standards of normality against which significant deviations in morphogenesis, growth rates, and morphology could be recognized and assessed. Once this is achieved it would be a logical step to determine if such deviations occurred in higher frequency and to a greater degree in populations affected with such clinical entities as anencephaly, cleft lip and/or palate, microcephaly, etc. This was pointed out by Kraus (*13*) in his study of calcification patterns and sequence as follows: 'We intend to . . . determine if significant deviations from this developmental pattern can be correlated with known fetal pathology. There are already promising hints in this direction in our preliminary surveys.' In a later paper outlining areas of research in dental genetics, Kraus (*14*) stated:

> . . . it seems evident . . . that the pattern of occlusal crown calcification for the mandibular first and second primary molars is under separate genetic control. If this is so, then the discovery in late-term fetuses of significant deviations from the established normal range of patterns might be indicative of genetic disturbance. On the other hand, the possibility of environmental intervention at a critical point cannot be ruled out. Currently we are examining fetuses of known abnormalities to determine if calcification pattern deviations are correlated. Like other well-known dental abnormalities, calcification pattern irregularities may be syndromic.

The present study represents the first such examination of an abnormal population, namely, one afflicted with cleft lip and/or palate. Its purpose was to determine if there is a significantly higher frequency of dental malformations in such a population as compared with a non-cleft population.

Dr. Jordan is Research Associate, Cleft Palate Research Center, and Head of the Department of Operative Dentistry, Dr. Kraus is Director of and Dr. Neptune is Research Assistant in the Cleft Palate Research Center, University of Pittsburgh.

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The present paper represents the first such report of the occurrence of morphological aberrations in the dental crowns of fetuses, children, and young adults afflicted with cleft lip and/or palate. Previous reports on dental irregularities in this type of population have been concerned primarily with number and malposition of teeth, with scant attention paid to the morphological features of the crowns themselves.

Goethe (*9*) described a suture of the hard palate which, anatomically, extended from the incisive foramen to the region between lateral incisor and cuspid. Goethe concluded that the area anterior to this line comprised the intermaxillary bone and that alveolar clefts result when the intermaxillary bone fails to unite with superior maxillary elements.

Albrecht (*1*) correlated observations made on animals and humans and concluded that the cleft was *not* between the intermaxilla and superior maxilla. He believed that the intermaxillary bone in its early developmental stages actually consisted of '4 os intermaxillaires' with two separate osseous segments on each side of the midline. According to his theory, the mesial segment, or endognathion, normally fused with the distal segment (mesognathion) and failure of these components to unite gave rise to clefts of the alveolar ridge (open endomesognathic suture).

Lucas (*18*) called attention to the familial incidence of cleft lip and cleft palate. In particular, he proposed that congenital absence of a maxillary lateral incisor be regarded as an abnormality closely related to cleft lip and cleft palate. His material was based largely on longitudinal studies of three individuals, each of whom showed a congenitally missing maxillary lateral incisor and each of whom fostered offspring with ipsilateral missing lateral incisor, cleft lip, or cleft lip and palate. He concluded that '. . . congenital absence of an upper lateral incisor may foretell the probability of cleft palate and hare-lip in a succeeding generation.' (His conclusion appears consistent with the theory of 'microforms' of clefts.)

Recently, Woolf and associates (*25*), in a study of the occurrence of lateral incisor 'anomalies' (by which they apparently meant absence of the tooth), examined the parents and siblings of 142 patients with cleft lip and/or palate, and determined that the frequency of this so-called anomaly was almost the same as in a noncleft control group consisting of 187 families. They concluded that a missing lateral incisor is not a microform of cleft lip and/or palate.

Turner (*23*) attempted to test Albrecht's theories by examining cleft palate casts. While such examinations allowed accurate evaluation of the relationship of teeth to the cleft, they did not '. . . enable one to state with absolute precision the particular part of the jaw in which the alveolar fissure is situated, and still less do they permit one to determine if a maxillo-intermaxillary suture coexists with the alveolar cleft.' In twelve of the fifteen casts examined, Turner observed a tooth (Albrecht's 'precanine') in the area between cuspid and alveolar cleft, a

point of some consequence because, if the intermaxillary bone supports the incisor teeth as theorized, and if Goethe's theory of failure of intermaxillary-maxillary union is valid, then the lateral incisor should always be found mesial to the cleft. In the same year, Coles (*4*), following the example of Albrecht and Turner, examined 31 cleft casts including patients with 'left-sided fissured alveoli, right-sided fissured alveoli, and double alveolar fissure.' Although the sample was small, Coles emphasized that there was not a single instance of fissure of the alveoli occurring between a true lateral incisor and canine on either side of the mouth nor was there sufficient evidence in any case of an increase in the number of teeth in the precanine region; there was distinct evidence, however, in some cases of the reverse condition and also of 'imperfect development.' In unilateral fissures, the maxillary central incisor was missing in 13 of 27 cases and, in 11 of 31 cases, a precanine was observed.

Warnekros (*24*), quoted in Millhon and Stafne (*20*), observed supernumerary teeth in patients with cleft palate and believed that this dental abnormality affected embryonic development in such a way that clefts of the lip and palate occurred.

Federspiel (*5*) emphasized the frequency with which lateral incisors were absent in cleft palate patients. He presented evidence to strengthen Albrecht's theory that the intermaxillary bone develops from four centers which fuse and later unite with the maxilla proper. Federspiel's interpretation of Albrecht's theory of development as it relates to the lateral incisor has some pertinence:

> At each point where the tooth is going to be formed, a bell shaped thickness appears at about the forty-eighth day or about the seventh week. It is interesting to know that about that time the union of the endo-, meso-, and exognathion should be united. If the mesognathion is delayed in its formation, the exognathion and endognathion may grow towards each other and prevent the full development of the mesognathion. The result is that the epithelial thickness to form a lateral tooth will be retarded, or arrested in its growth, or it may be obliterated.

Kirkham (*11*), while agreeing that '... it is obvious that this tooth (the maxillary lateral incisor) shows more embryologic irregularity than all other teeth,' emphasized that in unoperated clefts, there '... is invariably *no more disturbance in dentition than in the normal mouth of similar age*, except ... *in the region of the lateral incisor*.' (Italics ours) In operated cases, however, Kirkham considered that the percentage of missing teeth, misplaced teeth, and caries was greater than in unoperated cases. He observed that the cleft alveolus '... shows little or no growth disturbance except at the site of the cleft, namely the mesognathion.' He concluded that *there is no particular reason why dentition outside the cleft area should be disturbed.*

Millhon and Stafne (*20*) conducted a study to determine the incidence

of supernumerary and congenitally missing maxillary lateral incisors in cleft lip and cleft palate patients. Their series consisted of 81 cases in which the dental history revealed no extractions. The dentition of all patients was studied roentgenographically and, in certain cases, findings at surgery were presented. The authors concluded that supernumerary teeth are more common in individuals with cleft palate than in those with cleft lip or in cases seen at routine examination, and that the tendency for supernumerary primary teeth far exceeds the tendency for congenital absence. They too felt that supernumerary teeth were 'the result of some disturbance of the mesognathion at the time of development, which affects the developing tooth bud' in such a way as to produce splitting and consequent supernumerary teeth, obliteration of a tooth, or a markedly malformed tooth. These authors suggest that the mesognathion is more likely to be malformed than is the endognathion because of its position and smaller size.

Pruzansky (*21*) pointed out that the 'relation between degree of cleft in the alveolar process and the defect in the deciduous and permanent dentition is interesting,' adding that such defect might comprise abnormal number of teeth, irregularity in shape and structure, or malposition in the dental arch. Only one morphological 'defect' was mentioned, however, in connection with the deciduous lateral incisor which 'may be T-shaped or otherwise misshapen and generally malposed in the line of occlusion.' Pruzansky did not discuss tooth malformations in clefts other than those of the alveolar process.

McMillan (*19*) designed an interesting study in which he attempted to use dental developmental defects as a guide to the identification of insults producing defects in other parts of the body. In his study of 286 subjects with primary dentitions, he found a positive association between enamel hypoplasia and cerebral palsy, and between enamel hypoplasia and ocular fibroplasia. McMillan, however, did not deal specifically with cleft patients.

Spitzer (*22*), in a study of the dentition of 'mental defectives,' found dental abnormalities of rather frequent occurrence in mongoloids, but generally a normal dental morphology in microcephalics. He summed up his observations regarding mongoloid dentition as follows:

> The teeth are stunted, their crowns cone-shaped in the anterior and occlusally tapered in the posterior regions. Generalized microdontia and often partial anodontia occurring bilaterally and not infrequently involving all four quadrants are associated findings. The eruption of the permanent teeth is susceptible to abnormal delays.

The most recent study of dental anomalies found in cleft lip and palate was undertaken by Böhn (*2*) in 1963. Böhn studied 339 Danish children between the ages of three and seven years, all of whom had some form of cleft lip and/or palate. His primary interest was to determine the relative incidence of the numerical variants in the cleft

area in both dentitions and their interdependence and possible relation to type or degree of cleft, facial side, and sex. He was also interested in determining whether hypodontia outside the cleft area occurred as a typical feature of the disorder in any form of cleft. In addition to numerous dental measurements, he also dealt with various types of fissural teeth in both dentitions and the conditions under which the different types occurred, although in general Böhn was not particularly concerned with crown morphology. Of special interest, however, to the present study is his conclusion regarding the lateral incisor:

> ... a normal lateral incisor may not only consist of both a premaxillary and a maxillary component, but also possibly of only one of them if the other is missing. Under certain conditions the components show different form-giving capacity. Their growth potential is easily influenced, positively or negatively, by developmental disturbances in the primary palate region.

Böhn also found in confirmation of what Millhon and Stafne (*20*) had previously pointed out, that supernumerary teeth in the cleft area were more common in the primary dentition, whereas missing teeth in the cleft area were more common in the permanent dentition. He found that neither sex nor side of cleft did significantly influence the numerical variations. In addition, he observed that missing teeth outside the cleft region were never observed in primary dentitions, but that they were of frequent occurrence in the permanent dentitions.

In summary, investigations to date have focused primarily upon absent, supernumerary, or malpositioned teeth in cleft lip and palate subjects. Abnormal crown morphology has been reported only for the maxillary lateral incisors. The opinion was expressed that crown malformation need not be expected to occur outside the immediate region of the cleft. In view, however, of the proven frequency of external (*15*) and visceral malformations (*12*) in association with cleft lip and/or palate human fetuses, the present investigators felt that a specific study of the subject was warranted.

Sample and Methodology

Two types of populations were sampled. One sample consisted of 10 human cleft lip and/or palate fetuses. The other sample was made up of maxillary and mandibular dental casts of 192 individuals. The latter were divided into two groups: those with cleft lip and/or palate (N = 105), and those with normal palates (N = 87). Over 800 noncleft human fetuses served as a control sample. These have been reported by Kraus and Jordan (*16*). The sex distribution of the three fetal and postnatal cleft samples is presented in Table 1. The postnatal cleft sample represents cases treated in the private practice of Dr. Cameron Metz of Pittsburgh, Pennsylvania, and his very kind cooperation is hereby gratefully acknowledged.

TABLE 1. Sex distribution, where known, of 202 cleft and noncleft subjects.

Type of subject	*Male*	*Female*	*Sex unknown*	*Totals*
Postnatal cleft	65	37	3	105
Postnatal noncleft	—	—	87	87
Cleft fetuses	5	2	3	10
Totals	70	39	93	202

The noncleft control casts were selected from the Kraus collection and consist of white and Indian citizens of Arizona. The classification of the postnatal and prenatal cleft samples, classified according to type of cleft, is presented in Table 2. A four-fold classification is used based upon the topographical extent of the cleft (lip only, lip and alveolar ridge, lip and palate, and palate only). In Table 3 a reclassification is presented, based upon the number of cases in which the alveolar ridge was involved, regardless of other structures (lip and/or palate). In the postnatal cleft sample, 65% had involvement of the alveolar ridge. In the fetal sample, 70% had such involvement.

TABLE 2. Distribution of postnatal and prenatal cleft subjects according to type of cleft.

Type of cleft	*Postnatal*	*Prenatal*
Lip only	17	0
Lip and alveolar ridge	5	2
Lip and palate	64	5
Palate only	15	3
Other	4	0
Totals	105	10

TABLE 3. Distribution of postnatal and prenatal cleft subjects according to involvement of alveolar ridge.

Type of cleft	*Postnatal*	*Prenatal*	*Totals*
Lip only	17	0	17
Lip and alveolar ridge (with or without palatal involvement)	69	7	76
Palate only	15	3	18
Other	4	0	4
Totals	105	10	115

TABLE 4. Age distribution of postnatal subjects, according to type of dentition and chronological age.

Type of Dentition	*Age range*	*Cleft*	*Noncleft*
Primary	3 to 6 yrs.	21	13
Mixed	7 to 11 yrs.	57	47
Permanent	12 yrs. and older	27	27
		105	87

The tooth buds of the fetuses were recovered intact from the alveolar ridges and stained with alizarin red S. The follicles were then removed and the buds inspected under a dissecting microscope. Polaroid photographs were taken so that each bud could be compared with the buds of noncleft fetuses.

The ages of the 10 fetuses were as follows: one fetus at each of the ages of 10 weeks, 11 weeks, 12 weeks, 14 weeks, 18 weeks, 22 weeks, 25 weeks, and three fetuses at the age of 40 weeks. The age distribution of the postnatal subjects, both according to type of dentition and chronological age, is shown in Table 4.

The teeth were examined individually in the fetuses and both individually and collectively in the casts. Particular attention was paid to crown morphology, but number and position of the teeth in the dental arch were also observed. There were 15 different types of abnormalities identified in both cleft and noncleft subjects. These are listed in Table 5. Thirteen of the traits pertain to morphological irregularities of the crown, while two are concerned with the number of teeth

TABLE 5. Description of the 15 traits observed.

Trait Number	*Description*
1	Thick curved maxillary incisors
2	Excess mammelons
3	Exaggerated mammelons
4	Supernumerary teeth
5	Missing teeth
6	Peg-shaped incisors
7	T-shaped lateral incisors
8	Malformed mandibular first primary molars
9	Malformed maxillary first primary molars
10	Missing hypocone
11	Reduced hypocone
12	Fused protocone and metacone
13	Malformed mandibular bicuspids, a—1st; b—2nd
14	Irregular mammelons
15	Labial tubercles

present (supernumerary or missing teeth). These traits will be discussed in detail in the following section.

Results: Descriptive Analysis

A total of 15 separate and distinct dental abnormalities were observed with varying frequency in cleft individuals. The distribution of the various abnormalities by age and type of cleft for the prenatal and postnatal cleft populations will be discussed in the following section. It is of interest to note that many specimens in both samples displayed multiple abnormalities of both maxillary and mandibular dentitions, while others showed either single aberrations or in some cases none whatsoever. Of particular interest is the fact that, in prenatal cleft specimens below the age of 12 weeks, no deviations were apparent since at this time the teeth are in such an early stage of development that abnormalities cannot be detected by gross observation. The following description of the various traits is therefore based upon those specimens over 12 weeks of age.

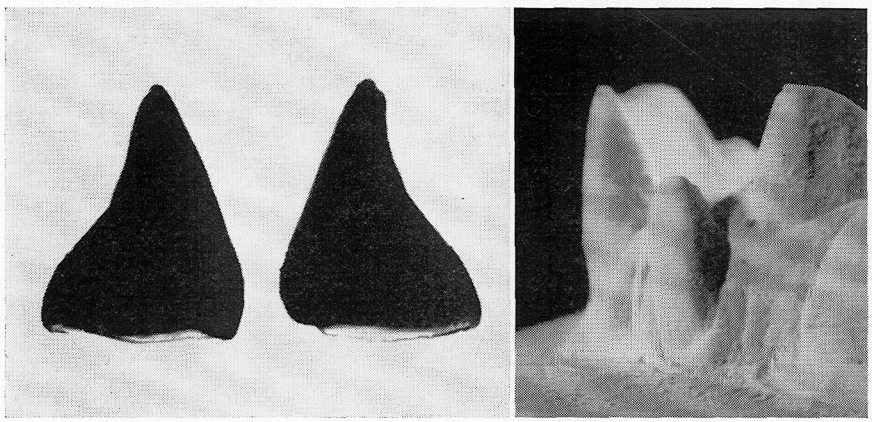
TRAIT #1: THICK CURVED CENTRAL INCISORS. The labial profile of the maxillary central incisor of a 36-week normal fetus shows only a slight convexity from the crest of curvature to incisal edge (Figure 1a). When viewed from the proximal aspect, the incisor crowns are usually wedge-shaped and taper cervico-incisally to form a relatively thin incisal edge. This typical outline is established for the primary incisors as early as 14 weeks *in utero* (Figure 2a). The primary central incisor of a cleft fetus at corresponding ages may show a more or less pronounced labial curvature with a thick lingually displaced incisal edge (Figures 1c and 2c).

This trait also occurs in the erupted dentitions of cleft individuals. Figures 1d and 2d show proximal and incisal views respectively of erupted maxillary central incisors of clefts. The pronounced curvature of the labial surface and thick incisal edge contrasts with the corresponding features of normal specimens (Figures 1b and 2b).

TRAIT #2: EXCESS MAMMELONS. As early as 14 weeks *in utero* primary incisor crowns show distinctive morphological features which depend to some degree upon the number, arrangement, and relative prominence of the mammelons which make up their incisal edges (*16*). The maxillary central incisor, for example, typically features three mammelons arranged in a distinctive pattern on the incisal edge (Figure 3a). The maxillary incisor of a cleft fetus (Figure 3c) may show as many as five or six mammelons distributed in an irregular manner, imparting to the incisal edge a more or less 'saw tooth' appearance. Similarly, the erupted permanent central incisors of cleft individuals may show excess or supernumerary mammelons (Figure 3d) in contrast to the usual number encountered in normal specimens (Figure 3b).

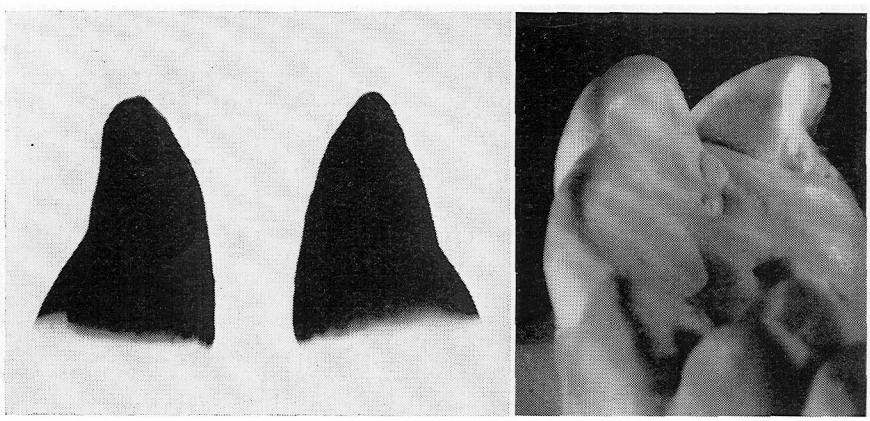
TRAIT #3: EXAGGERATED MAMMELONS. Mammelons are ordinarily seen as slight rounded eminences on the incisal edges of newly erupted

FIGURE 1. Trait #1: Thick curved central incisors



a) Maxillary central incisors, distal view 36 week fetus (noncleft)

b) Maxillary central incisors, distal view postnatal study model (noncleft)



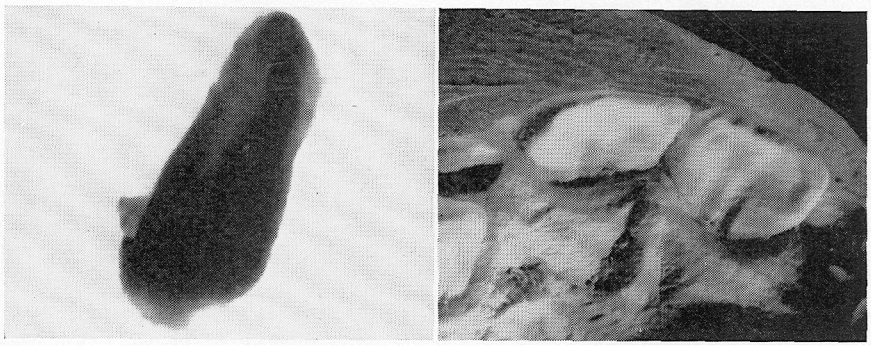
c) Maxillary central incisors, distal view 36 week cleft fetus

d) Maxillary central incisors, proximolinguo-incisal view postnatal study model (cleft)

anterior teeth (Figure 4a). The erupted permanent incisors of cleft individuals may show pronounced cusp-like elevations separated by distinct grooves (Figure 4b). This trait has not yet been observed in prenatal primary dentitions.

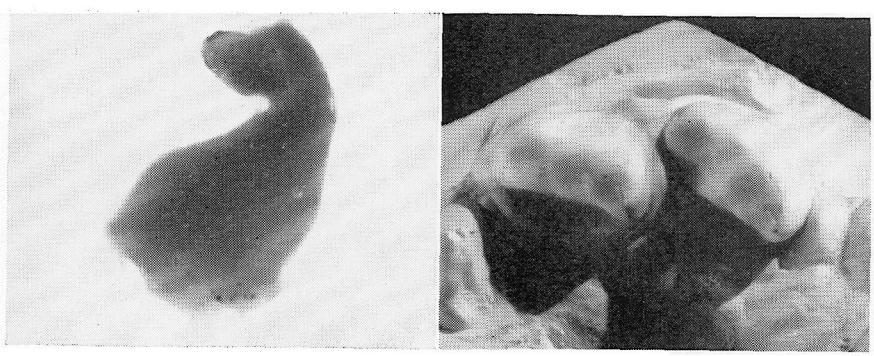
TRAIT #4: SUPERNUMERARY TEETH. Supernumerary teeth occur frequently in the lateral incisor region adjacent to an alveolar cleft (Figure 5d). Commonly, the morphology of the supernumerary tooth will vary depending on its positional relationship to the site of the cleft (*2*). The

FIGURE 2. Trait ※1 cont'd: Thick curved central incisors



a) Maxillary left central incisor, distal view 14 week fetus (*noncleft*)

b) Maxillary central incisors, linguo-incisal view postnatal study model (noncleft)



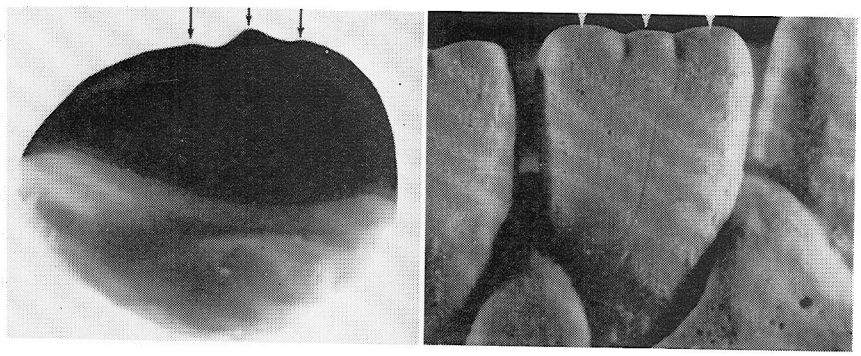
c) Maxillary left central incisor, distal view 14 week cleft fetus

d) Maxillary central incisors, incisal view postnatal study model (cleft)

left anterior dentition (Figure 5c) of a 25-week fetus affected with bilateral cleft lip, alveolar process and palate (see Figure 5a and b) shows two lateral incisors, (points 2 and 3 on Figure 5c) separated by the cleft. The incisor on the medial side of the cleft (point 2 on Figures 5b and 5c) exhibits a more or less typical lateral incisor morphology. Conversely, the morphology of the incisor distal to the cleft (points 3 on Figures 5b and 5c) is more irregular and approximates the form of the adjacent cuspid. It is impossible to determine, however, which is the supernumerary tooth. It is of interest to note that the central incisor of the same fetus exhibits the excess mammelons described under Trait ※2.

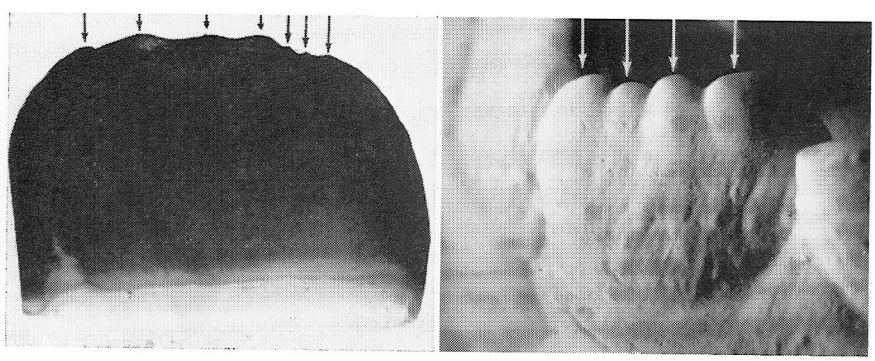
Trait ※5: Missing Teeth. Missing teeth in the area of the cleft occur in both primary and permanent dentitions, but it is much more common in the latter (*2*). In the analysis of cleft and noncleft casts any teeth not present in the dental arches at an age when such teeth would

FIGURE 3. Trait #2: Excess Mammelons



a) Maxillary right central incisor, lingual view 21 week fetus (noncleft)

b) Mandibular central incisor, labial view postnatal study model (noncleft)



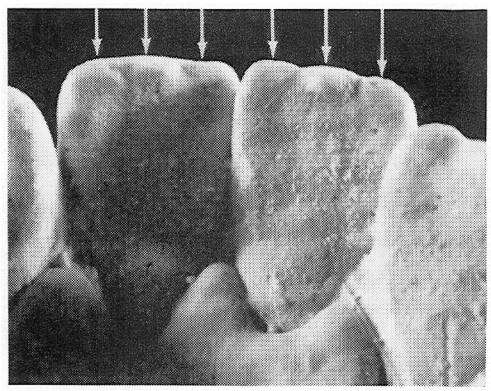
c) Maxillary right central incisor, lingual view 25 week cleft fetus

d) Maxillary right central incisor, labial view postnatal study model (cleft)

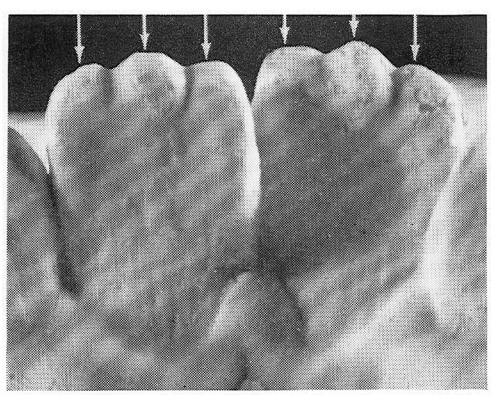
normally be expected were classified as 'missing.' Since unrestricted access to the dental arches of the fetal population was available, the absence of teeth could be more easily determined. For example, both maxillary central incisors were missing in the cleft fetus shown in Figure 6. Arrows indicate the approximate location of the right and left lateral incisors.

TRAIT #6: PEG-SHAPED INCISORS. Normally at 36 weeks *in utero*, maxillary lateral incisors present three distinct lobes which terminate incisally in their respective mammelons (Figure 7a). The middle or central lobe, the most conspicuous in terms of incisal prominence, is flanked respectively by a less prominent mesial lobe and a distal lobe which slopes cervically from the central lobe to end in a rounded disto-incisal shoulder. Incisor crowns (particularly laterals) showing a prominent central lobe with little or no apparent development of the mesial

FIGURE 4. Trait #3: Exaggerated Mammelons



a) Mandibular central incisors, lingual view postnatal study model (non-cleft)

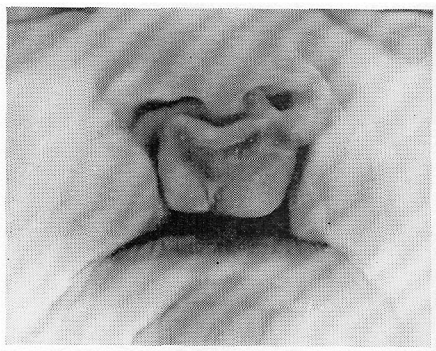


b) Mandibular central incisors, lingual view postnatal study model (cleft)

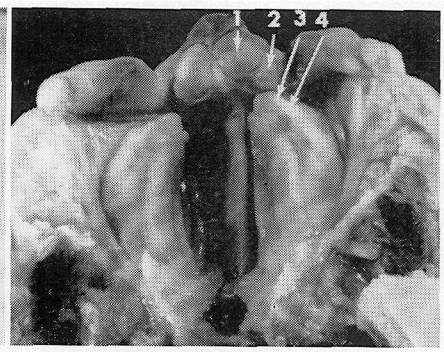
and distal lobes appear conical in outline and are generally referred to as 'peg-shaped.' Figure 7c shows the maxillary lateral incisors of a 36-week fetus affected with median cleft lip alveolar process and palate (see Figures 6a and 6b). It is of interest to note that although the left and right lateral incisors were located in corresponding positions adjacent to the cleft, they are not bilaterally symmetrical morphologically. Thus, the left incisor (L) is quite conical and shows no apparent development of either mesial or distal lobes, whereas the right incisor (R) shows some development of the mesial lobe and appears less peg-shaped.

Peg-shaped incisors may be a conspicuous feature of erupted cleft dentitons (Figure 7d) wherein they may feature a prominent lingual tubercle in addition to the apparent lack of development of mesial and

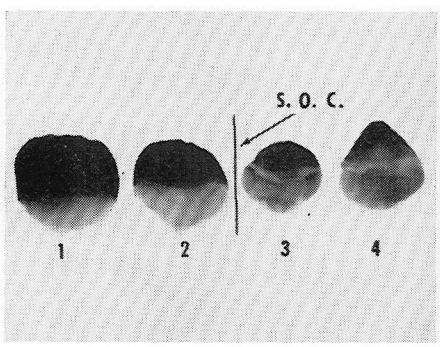
FIGURE 5. Trait ※4: Supernumerary teeth



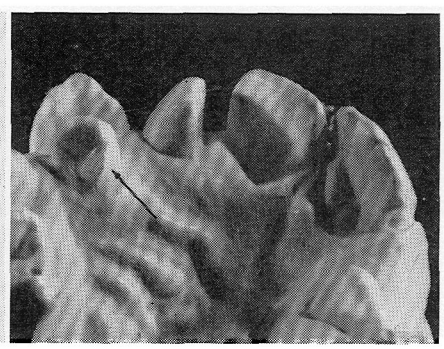
a) Bilateral cleft lip, alveolar process, and palate, frontal view
25 week fetus Specimen 4055



b) Palatal view of specimen 4055
Arrows indicate site from which teeth in fig. 5c were extracted



c) Maxillary left anterior teeth from specimen shown in fig. 5a and b. (Lingual view)
Vertical line s.o.c. indicates the site of cleft



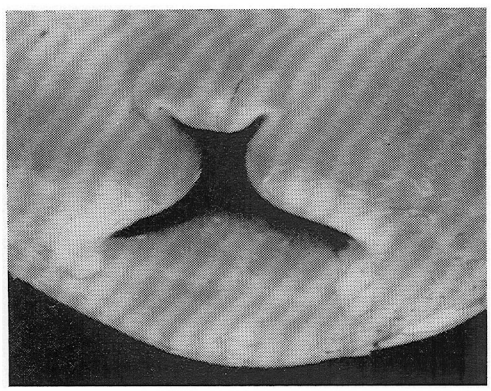
d) Maxillary right anterior teeth, lingual view postnatal study model (cleft). Note the presence of two lateral incisors

distal lobes, both features contrasting markedly with the normal morphology (Figure 7b).

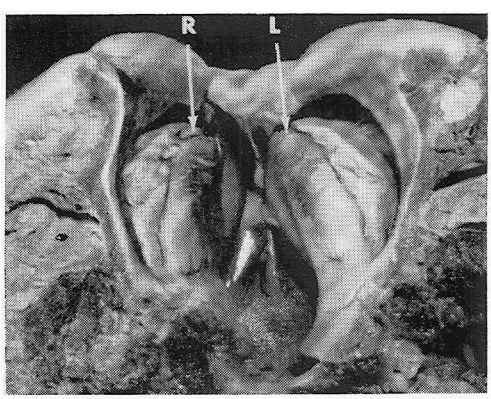
TRAIT ※7: T-SHAPED LATERAL INCISOR. A maxillary lateral incisor showing a prominent lingual tubercle connected to the incisal edge by a distinct ridge (Figure 8b) is designated as T-shaped (*3*). Although this trait has not yet been observed in prenatal cleft dentitions it is sometimes encountered in the erupted permanent incisors of cleft individuals. It is of interest to compare the relatively prominent lingual tubercle (L.T.) seen in both the peg-shaped trait (Figure 7d) and the T-shaped trait (Figure 8b).

TRAIT ※8: MALFORMED MANDIBULAR FIRST PRIMARY MOLAR. Generally the mandibular first primary molar at 36 weeks *in utero* is roughly

FIGURE 6. Trait #5: Missing Teeth



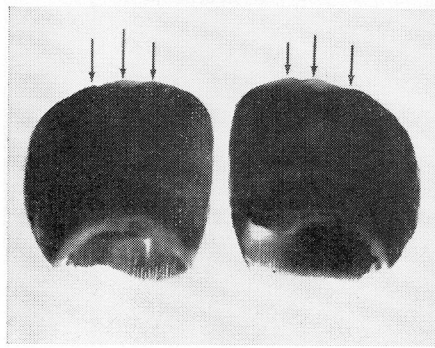
a) Median cleft lip, alveolar process, and palate, frontal view 36–40 week fetus W 371



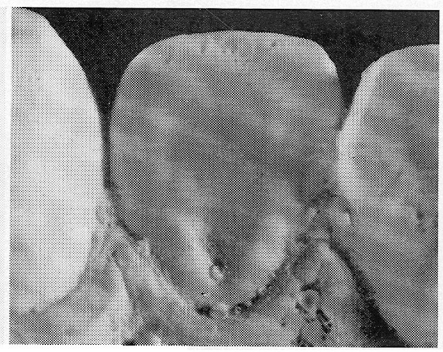
b) Palatal view of specimen W 371 Arrows indicate approximate location of maxillary lateral incisors. Both central incisors were missing

rectangular in outline as viewed from the occlusal (Figure 9a) and usually shows five cusps, occasionally six (tuberculum sextum) (Figures 9a and 9b). The protoconid (point 1) is the largest of the cusps in terms of height and basal width and occupies from ½ to ⅗ of the mesiodistal diameter of the crown. The metaconid (point 2), situated immediately lingual to the protoconid, is slightly smaller in dimension and presents a sharp conical form. The hypoconid (point 3) occupies from ⅓ to ⅖ of the buccal width of the crown and is sharply demarcated from the protoconid. The entoconid (point 4) is a small cusp situated on the distolingual corner of the crown and the hypoconulid (point 5), when present, is located on the distobuccal corner of the

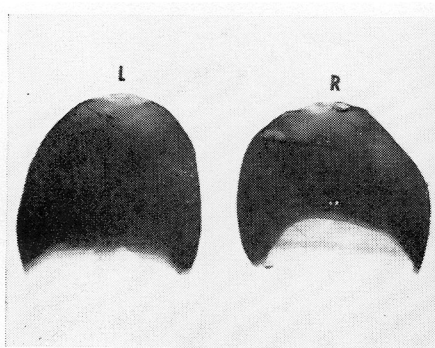
FIGURE 7. Trait #6: Peg-shaped incisors



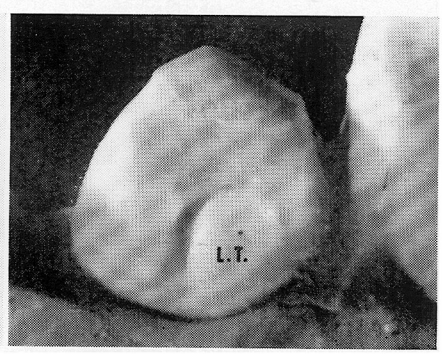
a) Maxillary right and left lateral incisors, lingual view 36 week fetus (noncleft)



b) Maxillary right lateral incisor, lingual view postnatal study model (noncleft)



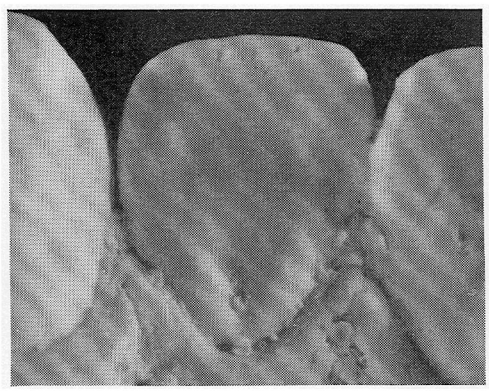
c) Maxillary right (R) and left (L) lateral incisors from cleft fetus shown in fig. 6 (lingual view)



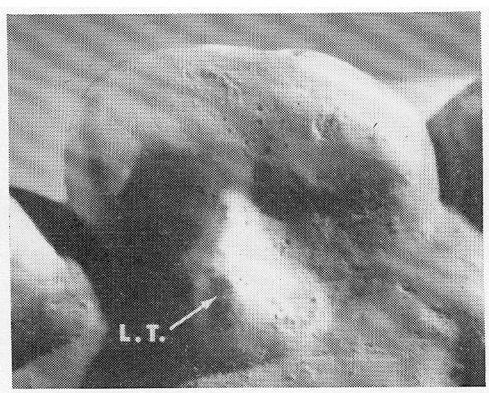
d) Maxillary right lateral incisor, lingual view postnatal study model (cleft). L.T.—lingual tubercle

crown immediately adjacent to the hypoconid. Not infrequently a sixth cusp (tuberculum sextum) is found on the distal marginal ridge between the hypoconulid and entoconid. The talonid cusps, namely the hypoconid (point 3), entoconid (point 4), and hypoconulid (point 5) customarily occupy the distal ⅔ of the occlusal surface and surround a deep talonid basin (*16*). Out of the total sample of 10 cleft fetuses four distinct deviations from this typical morphology were found. The first malformation occurred bilaterally in the mandibular molars (Figures 9c and 9d) of a 36-week cleft fetus (see also Figure 6). On occlusal view the crown presents a more or less triangular profile, tapering to an apex on the distolingual corner. In Figure 9 the hypoconid (point 3), displaced toward the distobuccal angle of the crown, is separated from the protoconid (point 1) by a deep furrow, shown as y on Figure 9c, on

FIGURE 8. Trait #7: T-shaped lateral incisors



a) Maxillary right lateral incisor, lingual view postnatal study model (noncleft)



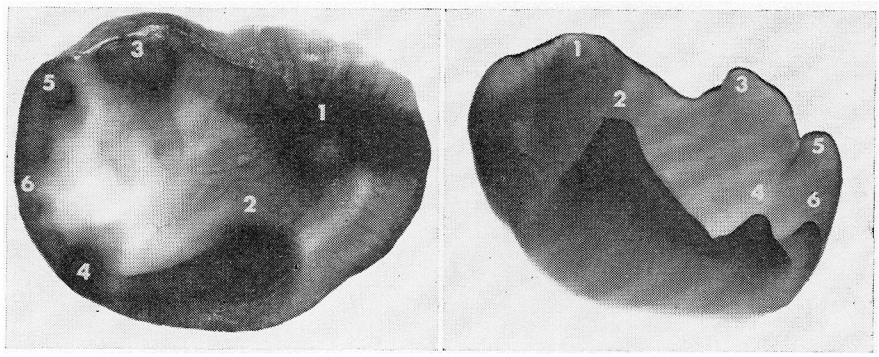
b) Maxillary left lateral incisor, lingual view postnatal study model (cleft). L.T.—lingual tubercle

the buccal surface. The entoconid (point 4) is missing and a pronounced U-shaped ridge extends distally from the protoconid (point 1) and curves mesially to terminate at the metaconid (point 2). An unidentifiable cusp, shown as x on Figure 9d, is situated on this ridge immediately distal to the protoconid.

The second deviant form, occurring bilaterally in the molars of a 36-week cleft fetus is marked primarily by the apparent absence of the hypoconid (Figure 10a). The protoconid (point 1) occupies the entire mesiodistal diameter of the buccal rim of the crown.

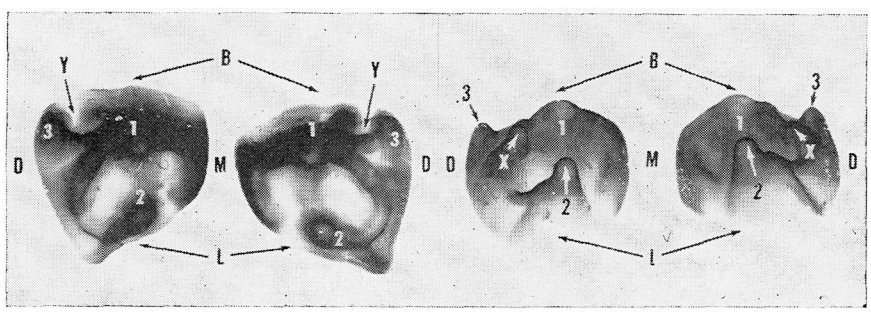
A bifid or double hypoconid (point 3) occurring bilaterally constitutes the major deviation in the lower molars of another 36-week cleft fetus (Figure 10c).

FIGURE 9. Trait #8: Malformed mandibular first primary molars



a) Mandibular left first primary molar, occlusal view, 36 week fetus (noncleft). 1—protoconid, 2—metaconid, 3—hypoconid, 4—entoconid, 5—hypoconulid, 6—tuberculum sextum

b) Mandibular right first primary molar, linguo-occlusal view, 36 week fetus (noncleft). 1—protoconid, 2—metaconid, 3—hypoconid, 4—entoconid, 5—hypoconulid, 6—tuberculum sextum

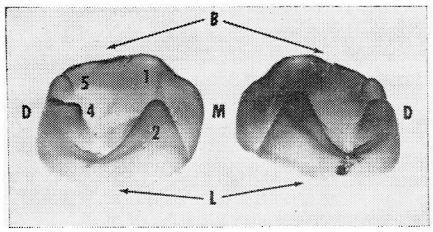


c) Mandibular left and right first primary molars, occlusal view, 36–40 week cleft fetus (W 371). B—Buccal, L—Lingual, M—Mesial, D—Distal. 1—protoconid, 2—metaconid, 3—hypoconid, y—furrow

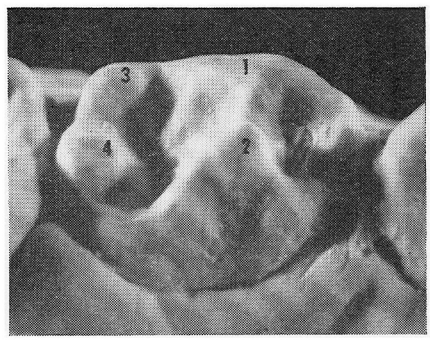
d) Same specimens as fig. 9c, linguo-occlusal view
x—accessory cusp

The fourth malformation is seen unilaterally in the left molar (Figure 10e) of a 25-week fetus with bilateral cleft lip alveolar process and palate (see also Figure 5a). The left molar crown, quite regular in oultine, shows a distinct buccal and distal placement of the hypoconid (point 3) and a deep furrow, shown as y, on the buccal surface separates this cusp from the adjacent protoconid (point 1). The entoconid (point 4, which lies almost in the center of the talonid basin), protoconid (point 1), and hypoconid (point 3) are linked by a highly atypical T-shaped ridge. The right molar of the same fetus shows a relatively normal morphology.

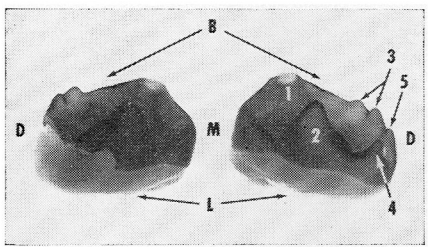
FIGURE 10. Trait #8: Malformed mandibular first primary molars



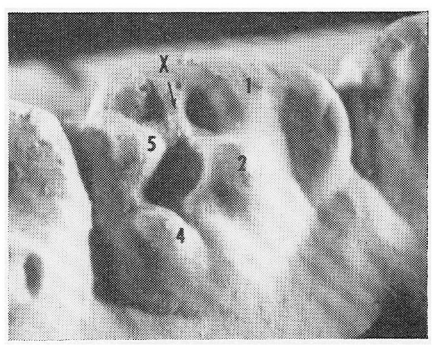
a) Mandibular left and right first primary molars, linguo-occlusal view, 36–40 week cleft fetus (4107)



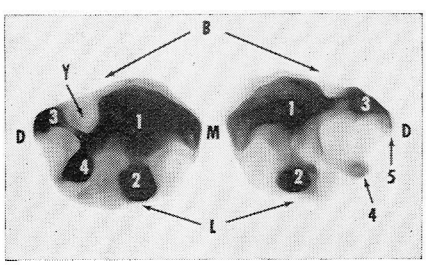
b) Mandibular left first primary molar, linguo-occlusal view, postnatal study model (noncleft)



c) Mandibular left and right first primary molars, linguo-occlusal view, 36–40 week cleft fetus



d) Mandibular first primary molar, linguo-occlusal view, postnatal study model (cleft)
x-Ridge



e) Mandibular left and right first primary molars, occlusal view, 25 week cleft fetus (4055)
y—furrow

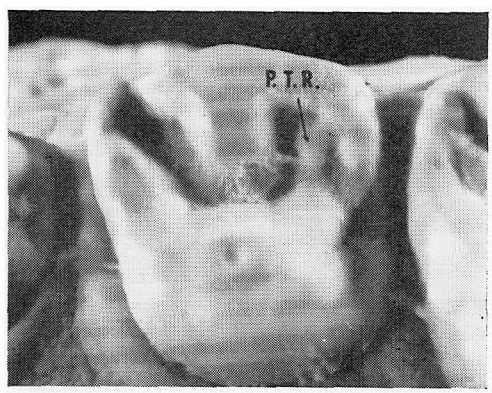
B—Buccal
L—Lingual
1—Protoconid
3—Hypoconid
5—Hypoconulid
M—Mesial
D—Distal
2—Metaconid
4—Entoconid

Malformations of the mandibular first primary molar are sometimes encountered in the erupted dentitions of clefts. Figure 10d shows a left primary molar (from a cleft specimen) which is marked primarily by the absence of the hypoconid (point 3). In addition, a prominent ridge, shown as x, coursing transversely across the talonid basin occupies the usual position of the buccal groove, and the entoconid (point 4) is displaced lingually. The occlusal outline of the crown is more or less triangular relative to the normal (Figure 10b).

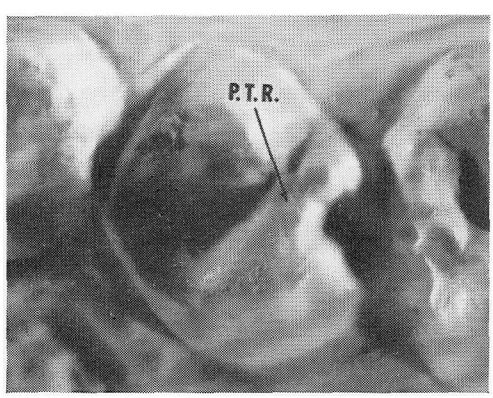
TRAIT #9: MALFORMED MAXILLARY FIRST PRIMARY MOLAR. The maxil-

lary first primary molar normally presents a roughly trapezoidal occlusal outline due to a slight lingual convergence of its proximal surfaces (Figure 11a). A faint 'posterior transverse ridge' (P.T.R.) is often present, crossing the distal portion of the occlusal surface between the metacone and protocone. A highly irregular deviant form (Figure 11b) occurs in the first primary molar of a cleft individual. The occlusal profile of the crown is more or less ovoid, and a pronounced transverse ridge (P.T.R.) crosses the occlusal surface to link the apices of the protocone and metacone. This trait has not yet been observed in prenatal dentitions.

FIGURE 11. Trait #9: Malformed maxillary first primary molars



a) Maxillary left first primary molar, linguo-occlusal view postnatal study model (noncleft)



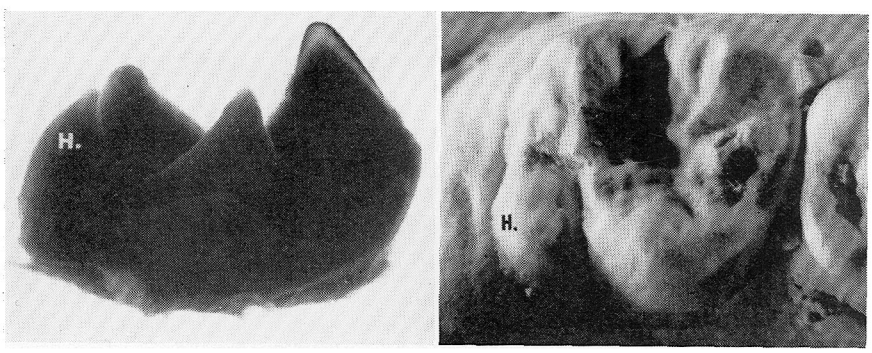
b) Maxillary left first primary molar, occlusal view, postnatal study model (cleft)

P.T.R.—Posterior Transverse Ridge

TRAIT #10: MISSING HYPOCONE. Maxillary second primary and first permanent molars characteristically present four main cusps. Prenatally the hypocone is the last of the major cusps to appear (point H, Figure 12a) and by the time of onset of calcification it is almost invariably a conspicuous feature of the normal embryonic crown (*16*). The hypocone may be missing entirely in prenatal cleft dentitions resulting in a tritubercular molar form (Figure 12c). Figure 12d shows the erupted maxillary second primary molar of a cleft individual. There is no apparent hypocone, and the distolingual groove which in normal maxillary molars (Figure 12b) separates the hypocone from the adjacent protocone, is absent.

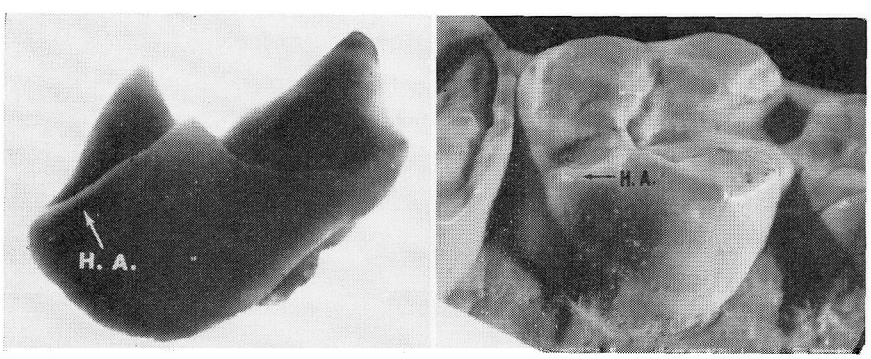
TRAIT #11: REDUCED HYPOCONE. Normally in prenatal dentitions the

FIGURE 12. Trait #10: Missing hypocone



a) Maxillary right first permanent molar, lingual view, 36 week fetus (noncleft)

b) Maxillary right second primary molar, occlusal view, postnatal study model (noncleft)

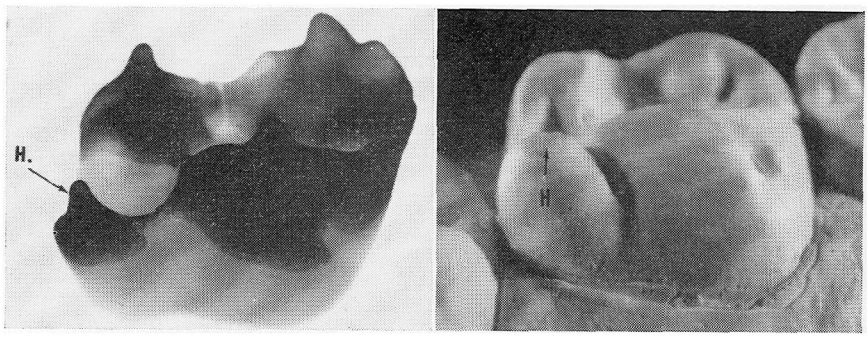


c) Maxillary right first permanent molar, lingual view, 36–40 week cleft fetus

d) Maxillary right second primary molar, linguo-occlusal view, postnatal study model (cleft)

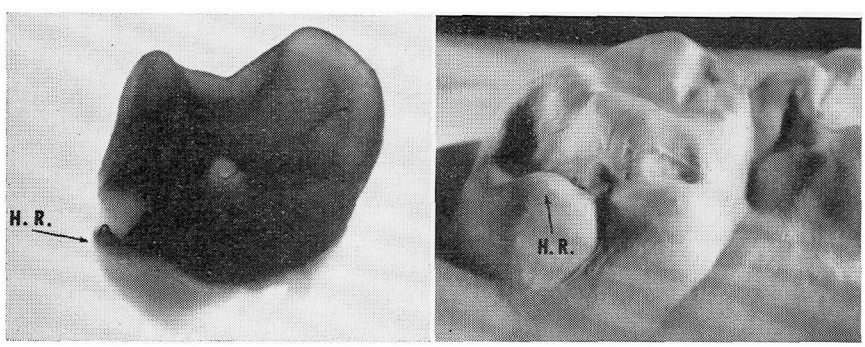
H.—Hypocone; H.A.—Hypocone Absent

FIGURE 13. Trait ⅋11: Reduced hypocone



a) Maxillary right second primary molar, linguo-occlusal view 32 week fetus (noncleft)

b) Maxillary right second primary molar, linguo-occlusal view postnatal study model (noncleft)



c) Maxillary right second primary molar, linguo-occlusal view 36–40 week cleft fetus W 371

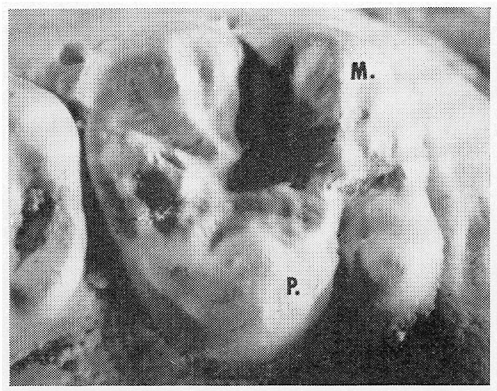
d) Maxillary right second primary molar, linguo-occlusal view postnatal study model (cleft)

H.—Hypocone; H.R.—Hypocone Reduced

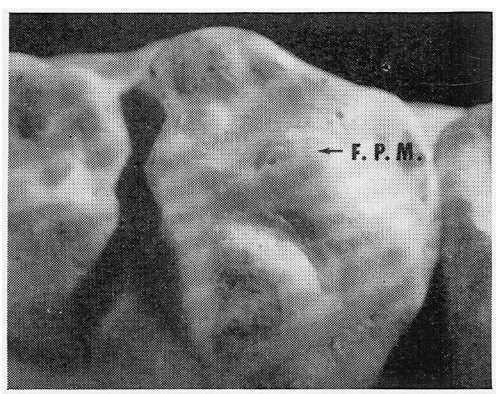
hypocone (point H, Figure 13a) attains a height of from ⅓ to ½ that of the adjacent protocone (*16*). The maxillary molar of a cleft fetus may show a hypocone considerably reduced in terms of size and prominence (Figure 13c). The reduction also occurs in the erupted molars of cleft individuals (Figure 13d) resulting in a noticeably altered crown pattern relative to the normal (Figure 13b).

Trait ⅋12: Fused Protocone and Metacone. Normally the protocone, shown as P., and metacone, shown as M., of maxillary second primary molars are prominent, well-defined elevations located at either extremity of the oblique ridge on the distobuccal and mesiolingual angles of the crown respectively (Figure 14a). This pattern is sometimes altered in the erupted molars of cleft individuals. A single cusp may

FIGURE 14. Trait #12: Fused protocone and metacone



a) Maxillary left second primary molar, linguo-occlusal view postnatal study model (noncleft)



b) Maxillary left second primary molar, linguo-occlusal view postnatal study model (cleft)

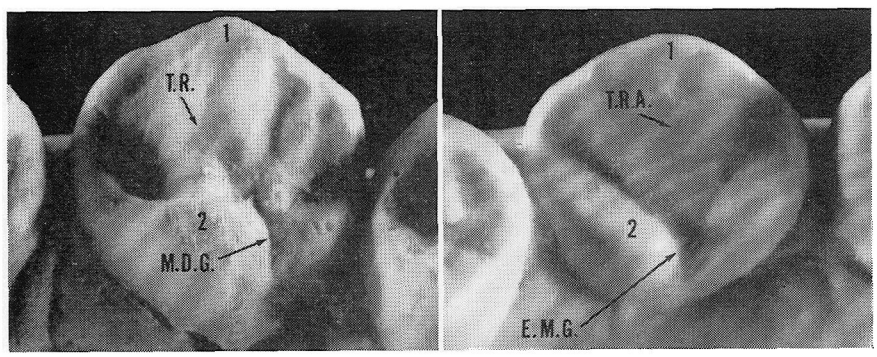
M.—Metacone; P.—Protocone; F.P.M.—Fused Protocone and Metacone

occupy the approximate midpoint of the oblique ridge in the apparent absence of protocone and metacone, shown as F.P.M. in Figure 14b. This relatively rare morphology is considered to result from a fusion of the two main cusps (*10*). This trait has not been encountered in prenatal dentitions.

Trait #13: Deviant Mandibular Bicuspids. The mandibular first bicuspid (Figure 15a) normally features two cusps; a strongly elevated buccal cusp (point 1), and a small lingual cusp (point 2). Pronounced central ridges course from the apices of both cusps toward the middle of

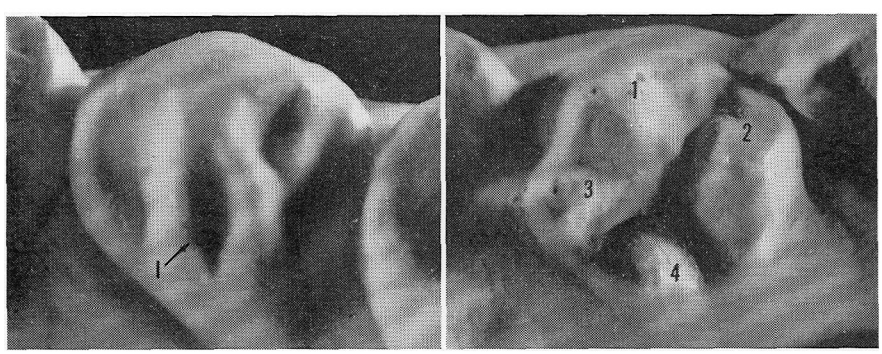
the occlusal surface, thereby forming a prominent transverse ridge (T.R.). The transverse ridge is interrupted by a central developmental groove which joins mesial and distal fossae. The mesiolingual developmental groove (M.D.G.) runs lingually from the central groove to interrupt the lingual rim of the crown. Three marked deviations from this typical morphology are found in the cleft series. One (Figure 15b) is distinguished by reduction of the lingual cusp (point 2), absence of the

FIGURE 15. Trait #13: Malformed mandibular bicuspids



a) Mandibular left first bicuspid, linguo-occlusal view postnatal study model (noncleft)

b) Mandibular left first biscuspid, linguo-occlusal view postnatal study model (cleft)



c) Mandibular right first bicuspid, linguo-occlusal view postnatal study model (cleft)

d) Mandibular right first bicuspid, occlusal view postnatal study model (cleft)

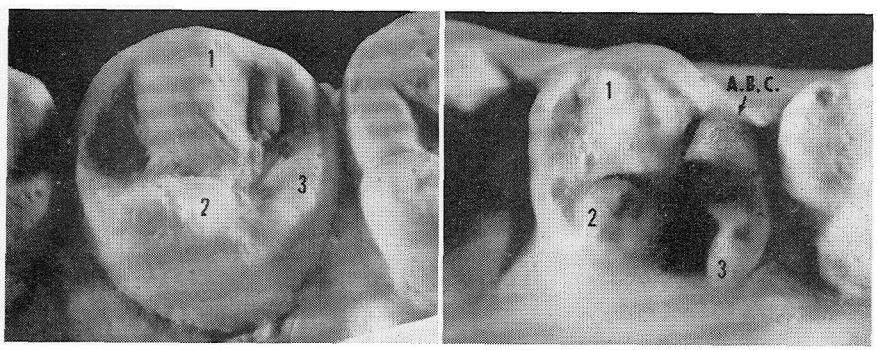
T.R.—Transverse Ridge
T.R.A.—Transverse Ridge Absent
M.D.G.—Mesiolingual Developmental Groove
E.M.G.—Exaggerated Mesiolingual Groove
i—Invagination

1—Buccal cusp
2—Lingual cusp
3—Accessory buccal cusp
4—Accessory lingual cusp

transverse ridge (T.R.A.) and a deep, exaggerated mesiolingual groove (E.M.G.). Another (Figure 15c) is marked by a deep invagination, shown as i, of the occlusal surface in that area normally occupied by the lingual cusp. The third (Figure 15d) features four cusps, two buccal (points 1 and 3) and two lingual (points 2 and 4), the latter approximately equalling the former in terms of size and prominence.

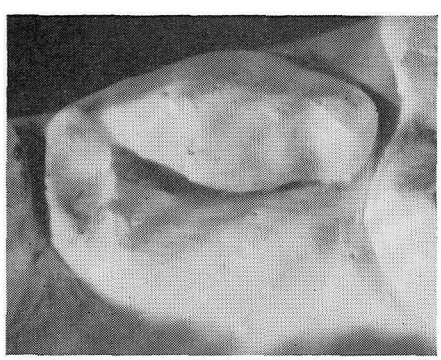
A typical mandibular second bicuspid (Figure 16a) usually features either two or three cusps. If three cusps are present—one buccal (point 1) and two lingual (points 2 and 3)—the occlusal profile is usually square; the two-cusp morphology exhibits a round occlusal profile. The

FIGURE 16. Trait #13 cont'd: Malformed mandibular bicuspids



a) Mandibular right second bicuspid, linguo-occlusal view postnatal study model (noncleft)

b) Mandibular right second bicuspid, linguo-occlusal view postnatal study model (cleft)



c) Mandibular right second bicuspid, occlusal view postnatal study model (cleft)

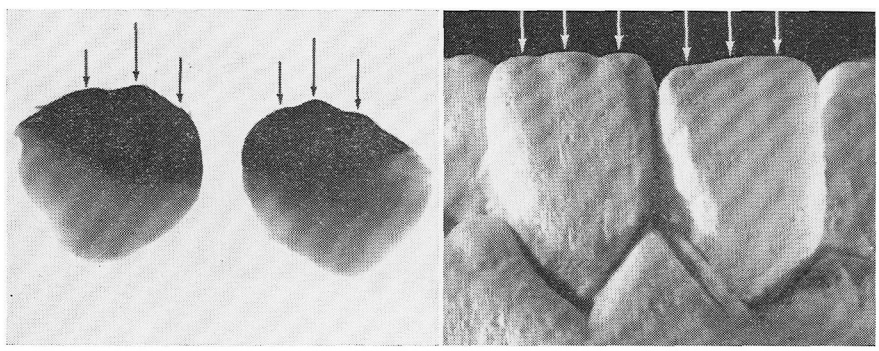
1—Buccal cusp
2—Mesiolingual cusp
3—Distolingual cusp
A.B.C.—Accessory buccal cusp

mandibular second bicuspid of cleft individuals may exhibit a molar-like morphology due to the presence of an accessory buccal cusp (A.B.C. in Figure 16b), or the crown may show a mesiodistally elongated or rectangular occlusal profile (Figure 16c).

Since mandibular bicuspids do not begin morphogenesis until after birth, this trait is not encountered in prenatal dentitions.

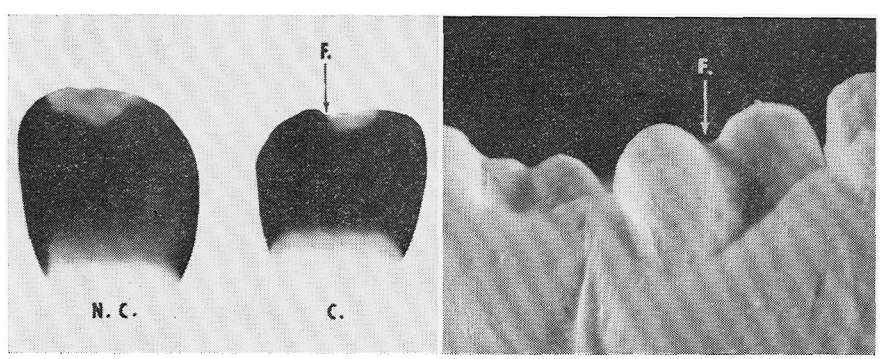
TRAIT #14: IRREGULAR MAMMELONS. The normal morphology of the incisal edges of fetal and newly erupted dentitions has been previously described (see Traits #2, 3 and 5). Typically, three mammelons are distinguishable on the incisal edges of normal lateral incisors during prenatal life (Figure 17a). Irregular development of mammelons may be

FIGURE 17. Trait #14: Irregular Mammelons



a) Maxillary right and left primary lateral incisors, lingual view 22 week fetus (noncleft)

b) Mandibular permanent central incisors, labial view postnatal study model (noncleft)



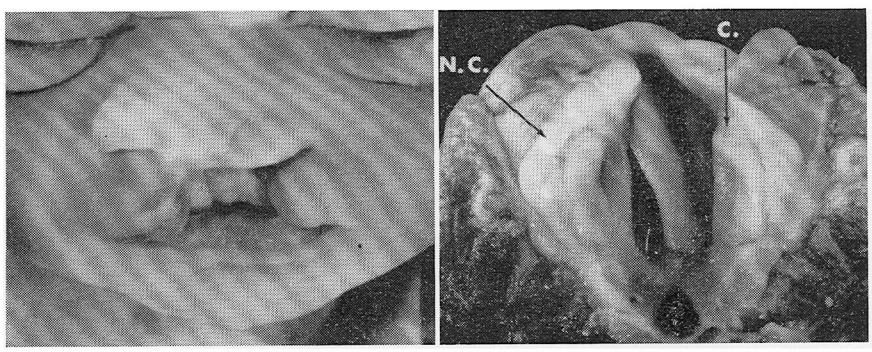
c) Maxillary right and left primary lateral incisors, lingual view 36–40 week cleft fetus

d) Mandibular permanent central incisor, labial view postnatal study model (cleft)

C.—Lateral incisor, cleft side
N.C.—Lateral incisor, noncleft side
F.—Incisal Fissure

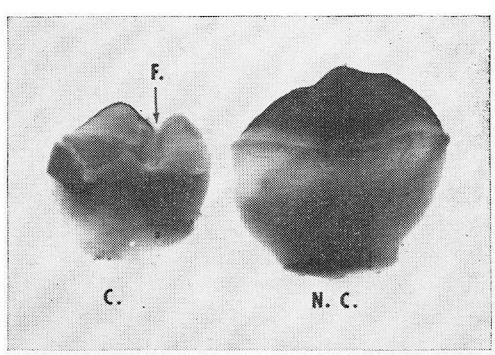
a conspicuous feature in the incisors of cleft fetuses. The malformation often takes the form of a pronounced fissure of the incisal edge especially in the lateral incisor immediately adjacent to the cleft. Figure 17c shows the lateral incisors of a 36-week fetus affected with a right unilateral cleft of the lip and alveolus. The lateral incisor from the cleft side, shown as c., shows a distinct fissuring (F.) of the incisal edge, while the contralateral tooth, n.c., appears normal morphologically. The lateral incisors of a 22-week cleft fetus (see Figures 18a and 18b) are shown in Figure 18c. The left incisor (shown as c. on Figures 18b and 18c) presents a pronounced fissure (F.) of the incisal edge, and is noticeably

FIGURE 18. Trait #14: Irregular Mammelons



a) Frontal view of left unilateral cleft lip, alveolar process, and palate 22 week fetus 4258

b) Palatal view of specimen 4258
C.—Position of lateral incisor on cleft side
N.C.—Position of lateral incisor on noncleft side



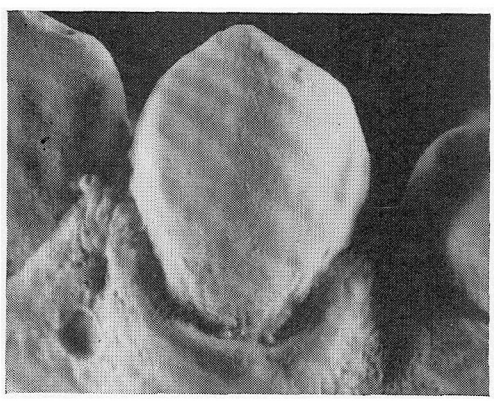
c) Maxillary right and left primary lateral incisors, lingual view, taken from specimen 4258
C.—Lateral incisor, cleft side
N.C.—Lateral incisor, noncleft side
F.—Incisal Fissure

smaller than its counterpart from the noncleft side (n.c. on Figures 18b and 18c).

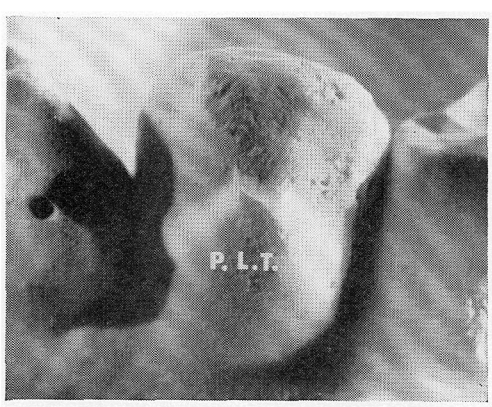
Irregular mammelons may also occur in erupted incisors remote from the site of the cleft. Figure 17d shows the *mandibular* left central incisor of a cleft individual with a pronounced fissure (F.) of the incisal edge. This imparts to the crown a bi-lobed appearance which contrasts with the normal three lobe morphology (Figure 17b). It is of interest to note that Böhn (*2*) reported the occurrence of this trait in the erupted lateral incisor of an apparently normal individual.

TRAIT #15: PARALABIAL TUBERCLES. Small cusp-like elevations on the buccal surfaces of maxillary and mandibular molars are referred to as

FIGURE 19. Trait #15: Labial tubercles



a) Mandibular left permanent cuspid, labial view postnatal study model (noncleft)



b) Mandibular left permanent cuspid, labial view postnatal study model (cleft)
P.L.T.—Para Labial Tubercle

paramolar tubercles (*3*). Cleft individuals may show similar structures on the labial surfaces of mandibular primary incisors and cuspids (P.L.T. on Figure 19b). Normally the labial contour of a primary anterior tooth is uninterrupted by accessory elevations (Figure 19a).

Results: Statistical Analysis

A tabulation of the frequency of abnormal traits occurring in the three study populations—cleft models, noncleft models, and cleft fetuses —is presented in Table 6. Of the 105 postnatal cleft dentitions examined through study models, 136 abnormal traits were observed. Traits 1 and 5 were the most frequent, together representing 49.2% of the total 136 abnormalities. The next most frequent was trait 6. The remaining traits each showed frequencies of less than 6%. In the 87 noncleft dentitions, only 15 abnormalities were discovered, nine of which comprised traits 2, 3, 5, and 8. Six cases were observed only once each. Five of the 15 traits did not occur. In the cleft fetal group, only eight specimens could be examined for the presence of dental abnormalities since in two cases the fetuses were too young to permit gross detection of the dental elements. In the eight fetuses, 24 dental abnormalities were observed, the most frequently occurring traits being 4, 6, 8, and 14, these four comprising 62.5% of the total observed. The orders of frequency of occurrence in the three populations are quite different (Table 7).

In the noncleft postnatal population, only 13 of the 87 individuals

TABLE 6. Frequency distribution of abnormal dental traits in cleft and noncleft study models and cleft fetuses.

Trait number	*Cleft models (N = 105)*		*Noncleft models (N = 87)*		*Cleft fetuses (N = 10)*	
	N	%	*N*	%	*N*	%
1	32	23.5	1	7.0	2	8.3
2	3	2.2	2	13.0	1	4.2
3	8	5.9	2	13.0	0	0.0
4	6	4.4	0	0	3	12.5
5	35	25.7	2	13.0	2	8.3
6	15	11.0	1	7.0	3	12.5
7	2	1.5	1	7.0	0	0.0
8	5	3.7	3	20.0	4	16.7
9	3	2.2	0	0	0	0.0
10	6	4.4	0	0	2	8.3
11	7	5.1	0	0	2	8.3
12	5	3.7	0	0	0	0.0
13	5	3.7	1	7.0	0	0.0
14	3	2.2	1	7.0	5	20.8
15	1	0.7	1	7.0	0	0.0
Totals	136		15		24	

TABLE 7. Order of frequency of traits for the three groups.

Order of frequency	*Cleft models*	*Noncleft models*	*Cleft fetuses*
1	5	8	14
2	1	2, 3, 5	8
3	6	1, 6, 7, 13, 14, 15	4, 6

showed abnormal dental traits, or a percentage of 14.9. In the cleft postnatal population, over half of the individuals were affected, or 54.3%. All of the cleft palate fetuses displayed abnormal dental traits (Table 8). In this respect there was a highly significant difference between the cleft and noncleft postnatal populations.

In Table 6, the total number of abnormal dental conditions observed in each of the three populations was reported. Dividing each by the total number of individuals in each population we obtain the average number of abnormalities per person. For the three populations these averages are: cleft models, 1.29; noncleft models, 0.17; and cleft feutses, 3.00. Application of a chi square test to cleft and noncleft model populations produces a value of 49.0 (Yates correction factor) and a P value of less than .0001. In other words, there is a highly significant difference in the average number of dental abnormalities per person between a cleft and a noncleft postnatal population. A statistical test is not necessary to show the striking difference between a cleft and noncleft fetal population. Examination of the dental buds of over 800 noncleft fetuses failed to reveal any of the dental abnormalities described in this report.

If the three populations are compared on the basis of the average number of abnormalities per affected individual, again statistically significant differences are found. The average numbers of abnormal dental traits per affected individual are: cleft models, 2.4; noncleft models, 1.1; and cleft fetuses, 3.0. A comparison of the three populations produces a chi square value of 9.1 with a P value of less than .02 (2 degrees of freedom). This indicates that an individual (pre- or postnatal) with a cleft who is affected with abnormal teeth is apt to have multiple abnormalities, whereas an individual so affected, but without a cleft, is likely to have but a single dental abnormality. Apparently, then, the

TABLE 8. Frequency of dentally abnormal individuals in total group. Chi square for cleft and noncleft models is 30.0, P is less than .0001.

Group	*Dentally abnormal*	*Dentally normal*	*Percentage of abnormal*
Cleft models (n = 105)	57	48	54.3
Noncleft models (n = 87)	13	74	14.9
Cleft fetuses (n = 8)	8	0	100.0

TABLE 9. Number of occurrences of single and multiple dental traits in cleft and noncleft individuals. Chi square is 23.9, P is less than .0001.

Subjects	*Single trait*	*Multiple traits*	*Total affected*
Postnatal clefts (n = 105)	11	46	57
Postnatal nonclefts (n = 87)	12	1	13
Fetal clefts (n = 8)	1	7	8
Totals	24	54	78

factor (or factors) producing the cleft has a definite bearing upon the number of dental abnormalities that occur.

In summary, it seems clear that not only are far more individuals in a cleft population affected with dental abnormalities than in a noncleft population, but that those individuals so affected tend to have multiple affected dental units rather than a single abnormality. This is demonstrated by Table 9, which shows the relative number of occurrences of single and multiple dental traits in the three populations under study. In the postnatal cleft population 81% of the dentally affected individuals show multiple dental abnormalities and 87% of the fetal cleft population have multiple dental traits. The postnatal noncleft population, on the other hand, revealed only one individual with multiple dental abnormalities out of 13 affected, or a frequency of only 7.7%.

A division of the postnatal cleft individuals according to type of cleft and presence or absence of dental abnormalities failed to reveal any significant differences (Table 10). The clefts were classified in terms of topographical involvement, in the assumption that cleft of the alveolar ridge (hence dental arch) would be more apt to be associated with dental abnormalities, whereas clefts of the lip or palate alone would not involve the dentition. A chi square test (Yates correction factor, 2 degrees of freedom) failed to produce a statistically significant value (P was greater than .10). This would appear to indicate that the area

TABLE 10. Frequency of dental abnormalities in cleft study models according to type of cleft. Two cases were unknown in each of the dental abnormalities groups. Chi square is 4.2, P is greater than .10.

Type of cleft	*Presence of dental abnormalities*	*Absence of dental abnormalities*	*Totals*
Lip only	10	7	17
Alveolar involvement	41	28	69
Palate only	4	11	15
Totals	55	46	101

affected by the cleft (lip alone, palate alone, or alveolus with either lip or palate or both) is not significantly correlated with the presence or absence of dental abnormalities. It is the *fact* of cleft, not the *nature* of the cleft, which is significantly involved with the appearance of abnormalities of dental morphology.

An important additional point emerges. One would assume, perhaps, that the mandibular teeth, being far removed from the cleft area, would not be affected. Such is not the case. Three of the 15 dental traits (8, 13, and 15) involve only the mandibular dentition and in the total cleft population (prenatal and postnatal) there were 15 cases observed (see Table 6). In four other traits (2, 3, 6, and 14), either maxillary or mandibular teeth were involved. There was no striking disparity in the frequency of occurrence of dental abnormalities in the two arches. On the other hand, the occurrence of supernumerary teeth or the absence of teeth was confined to the maxillary dentition and apparently was directly correlated with the physical fact of the cleft.

Discussion

The impact of Fogh-Andersen's work (*6*) on the thinking of investigators in the field of cleft palate research has been so striking that today there is almost universal acceptance of the major role of heredity as an etiological factor in the development of this malformation. In a later work (*7*) Fogh-Andersen reconfirmed his belief that heredity is *the main etiologic factor*, in the face of accumulating evidence that certain exogenous factors such as stress, nutritional deficiencies, infections, intoxications, etc. during the prenatal period must be considered. In his most recent publication (*8*) Fogh-Andersen confesses great doubt as to the relative importance of heredity and environment, although still adhering to the concept that heredity is *an important etiological factor*. Nevertheless he points out that cleft lip, whether alone or with cleft palate, has no 'genetic connection.' Furthermore he advises against any kind of drugs during the first months of pregnancy. On the other hand, Woolf, Woolf, and Broadbent (*26*), in a study of almost 60,000 live births in Salt Lake City, claim that both cleft lip and cleft lip with cleft palate have a genetic component in common.

Without denying the possible heterogeneity of etiological factors, including heredity, the new findings obtained by the study of human fetuses, both with and without clefts, would appear to mitigate the importance of the genetic component. The work of Kraus, Kitamura, and Ooe (*15*) and Kitamura and Kraus (*12*) clearly indicate the significantly frequent but unpredictable association of both external and visceral malformations in cleft palate human fetuses. There was no evidence of any fixed constellation of anatomical abnormalities associated with the cleft, regardless of type. Any organ or structure, or combination of several, could be affected, and to varying unpredictable degrees. The

present investigation, in which a significantly higher frequency of dental abnormalities accompanies the cleft, both in fetuses and in living individuals, strongly supports the previous work in emphasizing the nongenetic association of widely diffuse bodily abnormalities in cleft lip and/or palate populations. It is quite obvious that the picture is not that of an hereditary syndrome.

An hypothesis more consistent with the observations made in the investigations of these and the present authors would hold that the factor or factors responsible for the cleft are also operating, in an apparent haphazard manner, throughout the rest of the body. We are not prepared to attempt to identify these factors, but it seems obvious that they are already at work before the 47th day of prenatal life when the palatal shelves first begin to fuse (*17*). Viral or bacterial infection may well be a more important etiological factor than heredity, but only more detailed research can confirm or deny this possibility.

It should be pointed out that all of the 15 dental abnormalities reported in this paper have been observed in noncleft individuals. The difference is not qualitative but rather quantitative. This is precisely the case in both external and visceral malformations observed in cleft palate fetuses; the same conditions may be found in noncleft fetuses but with much less relative frequency. Furthermore, the occurrence of multiple abnormalities of teeth, external body structures, and visceral organs in a single individual is far more common in cleft than in noncleft subjects.

Of the five dental abnormalities that were not observed in the postnatal noncleft sample (see Table 6), four were recorded by Jørgensen (*10*) in a Danish noncleft population. The fifth (malformed maxillary first primary molar) was observed in three postnatal cleft subjects. In the cleft fetuses, six of the abnormal dental traits were not observed. This was in part due to the small sample size. One trait (malformed mandibular bicuspids) could not, of course, be observed in human fetuses since the bicuspids do not begin to form until after birth.

The occurrence of malformed teeth in the mandibular arch as well as in areas of the maxillary arch far removed from the site of the cleft is in direct opposition to Kirkham's statement (cited earlier) to the effect that teeth outside the cleft area are not expected to be disturbed.

Summary

The dental study models of 105 cleft and 87 noncleft individuals were examined and analyzed. In addition, the tooth buds of 10 human fetuses with cleft lip and/or palate were extracted, stained with alizarin red S, and compared with the tooth buds of approximately 800 noncleft human fetuses. Fifteen dental traits, 13 morphological and two numerical, were determined to be abnormal in terms of their frequency of occurrence. The distribution of these traits in the cleft and noncleft populations was noted

with the following results: a) In the postnatal cleft dentitions, 136 separate dental abnormalities were observed as compared with 15 such cases in the postnatal noncleft dentitions. b) In terms of the number of individuals affected in the postnatal cleft population, 54.3% showed one or more dental abnormalities, while only 14.9% were affected in the noncleft population. These differences were statistically highly significant. c) In the eight fetuses whose tooth buds were well enough developed to be grossly observed, all were affected, presenting a total of 24 abnormal dental traits. d) When the average number of abnormalities per individual is based upon *total* number in each sample, the results are as follows: cleft models, 1.29; noncleft models, 0.17; and cleft fetuses, 3.00. Again there is statistically a highly significant difference between nonclefts and clefts. e) In terms of the average number of abnormalities per *affected* individuals the results were as follows: cleft models, 2.4; noncleft models, 1.1; and cleft fetuses, 3.0. The averages for both postnatal and prenatal cleft subjects were significantly higher than for the noncleft subjects. f) In the postnatal cleft sample, 81% of the dentally affected individuals and 87% of the affected fetal clefts showed multiple dental abnormalities, whereas only one of the 13 postnatal noncleft dentally affected individuals revealed multiple dental abnormalities. g) There was no correlation between type of cleft and presence or absence of dental abnormalities. h) The 13 *morphological* dental abnormalities were found in both maxillary and mandibular dental arches, but supernumerary or absent teeth occurred only in the maxillary dentition.

It can be concluded that neither the cleft itself nor the type of cleft is an etiological factor in the occurrence of morphological abnormalities in the individual dental units. It would appear that the development of the dentition along with that of the other organs and structures of the body may be affected by the same etiological factor or factors that are responsible for the cleft lip and/or palate.

Cleft Palate Research Center
University of Pittsburgh
Pittsburgh, Pennsylvania 15213

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